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EFFECT OF GRAVITATIONAL OVERLOADS, HYPOKINESIA AND HYPODYNAMIA ON THE VESSELS OF THE PULMONARY BLOOD CIRCUIT

A. A. Kasimtsev

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EFFECT OF GRAVITATIONAL OVERLOADS, HYPOKINESIA AND HYPODYNAMIA ON THE VESSELS OF THE PULMONARY BLOOD CIRCUIT

A. A. Kasimtsev
Department of Normal Anatomy (Head: Prof. M. G. Prives, Honored Scientist),
First Leningrad Medical Institute im. I. P. Pavlova

The basic laws for morphological changes in the vessels of organs and tissues /82* affected by gravitational stress are found in the works of M. G. Prives (1963, 1968, 1970). In respect to morphological studies of the vessels of the pulmonary blood circuit the work done in this area (Yu. N. Korolev, 1963, 1965; I. S. Gil'bo, 1966; Glaister, 1968; V. V. Kupriyanov, 1969; V. G. Petrukhin, 1969; I. Z. Shtabrovskiy, 1971) does not present the whole picture of changes in pulmonary circulation due to overloads. Recently research has appeared, which indicates alterations in the neural apparatus of pulmonary circulation vessels and of the very wall of the pulmonary vessels under stress (S. I. Yevloyev, V. M. Klebanov and S. S. Mikhaylov, 1970; Ye. A. Dyskin, I. D. Lev and N. S. Shadrina, 1971; I. V. Shust et al., 1971).

Taking into account, that gravitational stress, hypokinesia and hypodynamia experienced in high altitude and space flight act in a determined sequence, we set up as our goal to study changes in the pulmonary blood vessels both under overload and under hypokinesia and hypokynamia and likewise to discover the changes occurring when there is a sequential combination of these two factors.

The research was done on 148 males rabbits weighing 2.0-2.5 kg. 15 animals were used to study the vessels of pulmonary circulation under normal conditions. 26 were subjected to single, prolonged threshold endurable gravitational overloads in the chest-back direction. For 117 animals there was simulation of hypokinesia and hypodynamia by keeping them in narrow cages for periods of 1-8 weeks. Of these animals 50 were killed after different periods, whereas 57, following hypokinesia and hypodynamia, were subjected to single, continuous threshold endurable chest-back stress. The pulmonary circulation vessels were studied by rentgenography and microrentgenography following injection of the vessels of the truncus pulmonalis and pulmonary

*Numbers in the margin indicate pagination in the foreign text.

vessels with Hauch's rentgenocontrast mass as modified by M. G. Prives, injection of the pulmonary circulation vessels with an India ink gelatin mass and Herot mass with subsequent preparation of 25-40 micron sections and clearing by the method of A. M. Malygin; likewise histologically by Van Gieson and hematoxylin-eosin staining.

Normally the truncus pulmonalis of the rabbit divides into two large arteries which enter the pulmonary hilus and follow a caudal course (Fig. 1). Arteries of the II order go out to the respective lobes in the direction of the lobar apex. Segmental arteries of the III order go out from the lobar arteries, etc. The rentgenograms distinctly show the injected arteries down to those of the VII order. Microscopically the pulmonary veins of the rabbit present the following appearance on the rentgenograms: the small veins of the VII-V orders mutually anastomose and fuse to form veins of the IV order that are subsegemental and follow a somewhat sinuous course. The joining of veins of the IV order forms larger segmental veins, etc. till the common right and left caudal and cranial veins have formed (Fig. 2) and empty into the left atrium. The pulmonary capillaries form a network interlacing the alveoli (Fig. 3); the course of the capillar- /83 ies is somewhat sinuous, their diameter varying from 8-12 microns. We were not successful in finding in the preparations the so-called "mainline capillaries" and "paracapillaries" that bypass the capillary network and have been noted by B. V. Ognev (1949), G. I. Mchedlishvili (1958) and Von D. Dscherov (1970).

1

Fig. 1. Pulmonary arteries of the right diaphragmatic lobe of the normal rabbit. Rentgenogram.

Effect of Transverse Stress. A study of preparations from animals subjected to overload showed, that arterial vessels of all orders become severely twisted (Fig. 4a), the overall number of injected arteries increases in the dorsal portions of the diaphragmatic lobes and in the radical zones due to the appearance of small reserve vessels which normally cannot be injected. Vessels of the larger orders are dilated and arteries in the peripheral portions of the pulmonary lobes are markedly constricted — vessels of the IV and V orders. Pulmonary veins of all orders are dilated and sinuous

as compared with normal (Fig. 4b). There is an increased number of injected small venous vessels in the dorsal portions of the diaphragmatic lobes. There is a significant increase in the total area of the capillary bed compared with normal. Capillary diameter varies from 10-12 μ to 20-25 μ and the course of the capillaries is more sinuous (Fig. 4c). There are varicose protrusions of the capillary wall. In some areas of pulmonary tissue there is a clear border around the vascular wall, evidence of symptoms of perivascular edema (Fig. 4d). In the histological preparations there was no sign of severely altered vascular walls in the form of tumid and crumbling layers, such as noted by Yu. I. Korolev (1965) dealing with single transverse overloads.

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Fig. 2. Pulmonary veins of normal rabbit. Rentgenogram.

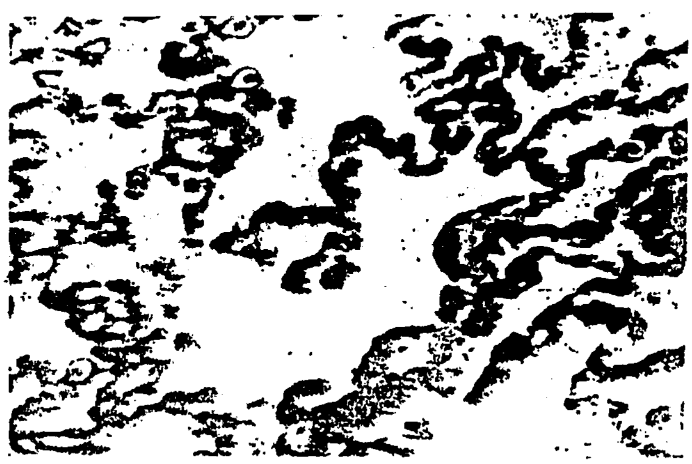

Fig. 3. Capillary bed of normal rabbit. India ink gelatin injection. Obj. 40, ocular 7.

It is possible to find many similarities, if one compares the chan-/84 ges described above in respect to the pulmonary blood circuit acted upon by single, threshold endurable chestback overloads and those observed by I. S. Gil'bo (1966) in the truncus pulmonalis system under the influence of headpelvis stress. The fact is borne out by the findings of V. G. Petrukhin (1969) showing, that the degree of pulmonary blood distribution is a function of the direction, and not of the magnitude, of the overload.

Thus the changes in the pulmonary blood vessels under the influence of stress are these: distal portions of III and IV order arteries in inferior lobes constricted; veins of all orders dilated; occurrence of sinuosity in arterial and venous vessels;

abnormally large number of small arterial and venous vessels in dorsal portions of inferior lobes due to opening of reserves; overall area of capillary bed notably enlarged, capillary wall deformed; greatest changes noted in dorsocaudal portions of lungs, especially in inferior lobes.

Dynamic Study of the Effects of Hypokinesia and Hypodynamia. After 1 and 2 weeks there was a macroscopic finding of some constriction in the distal portions of arteries of the IV order and dilation of small veins. The capillaries were slightly more dilated than normal (Fig. 5a) and sinuous. Along their course the lumen was uneven (from 8-10 μ to 15-17 μ). There was an abnormally large number of anastomoses between the system of pulmonary and bronchial arteries. After 4 weeks hypokinesia and hypodynamia the effect of these factors appeared in notable constriction of the distal portions of arteries of the III and IV orders as against the previous periods and in some parts of the lungs this was so pronounced, that the rentgenocontrast mass could not pass through the constricted vessels and foci of spasm appeared (Fig. 5b). Veins of all orders were even more constricted and sinuous and changes developed in the capillary bed.

After 6 and 8 weeks hypokinesia and hypodynamia the constricted arteries became even more severely constricted and the arterial network became very poor. The veins of all orders (F . 5c) appear extremely sinuous and markedly dilated. Under the influence of 8 weeks hypokinesia the capillary bed undergoes even greater changes (Fig. 5d): some of the capillaries are severely constricted to the point of a closed lumen, some dilated and deformed. the number of capillaries seen in various /87 parts of the pulmonary tissue is not constant, so that the area of the capillary bed does not remain constant.

Histologic study shows tumidity of the central membrane of the vascular wall of small arterial vessels during the early hypokinetic periods, but due to hypokinesia the 8th week markes the setting in of a reduction in the number of muscular fibers and an expansion of connective tissue. The membranes of the vascular wall are loosely juxtaposed, the adventitia tumid and its cells loosely distributed.

Thus the changes the pulmonary blood vessels affected by hypokinesia and hypodynamia are expressed as follows: arterial vessels of the III and IV orders in the distal portions are constricted; veins of all orders are dilated and venous vessels ap-

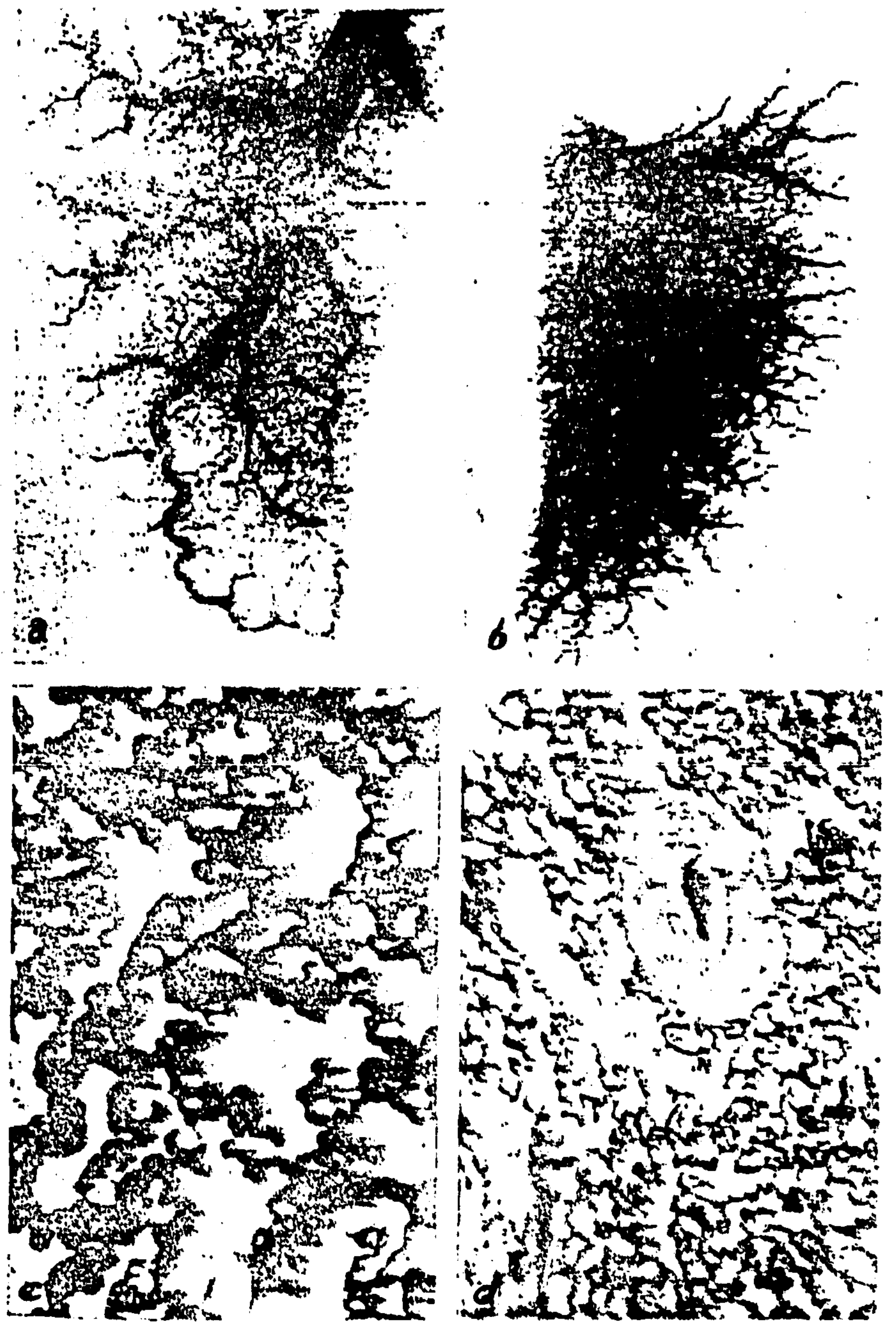

Fig. 4. a - pulmonary arteries affected by stress (left inferior lobe; rentgenogram); b - pulmonary veins following stress (left inferior lobe; rentgenogram); c - pulmonary capillary bed following stress; India ink gelatin injection; object. 40, ocular 7; d - perivascular edema; India ink gelatin injection, hematoxylin-eosin; objective 20, ocular 7.

pear sinuous; increase in total area of capillary bed due to dilation of capillaries; indicated changes develop in proportion to length of hypokinesia and hypodynamia.

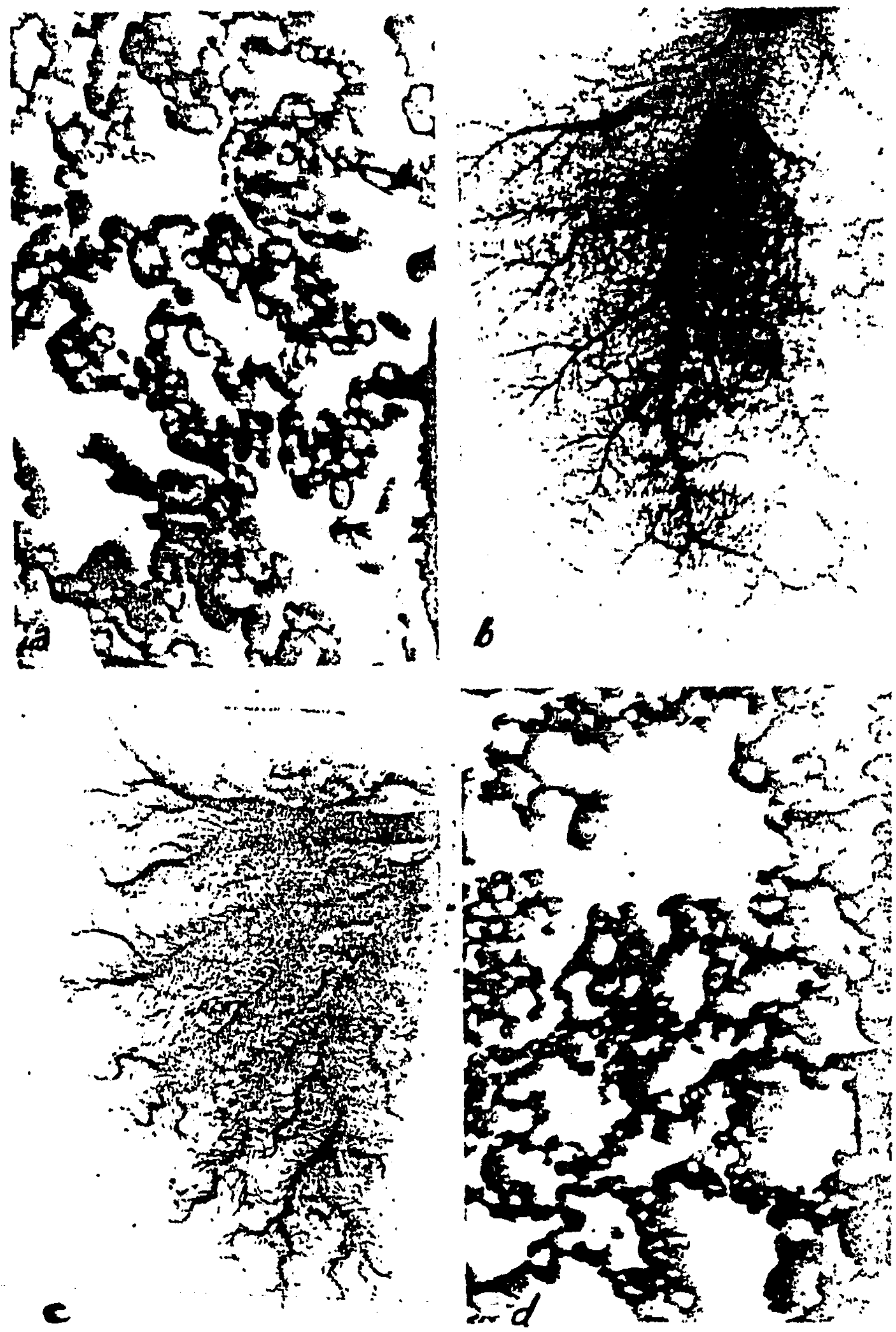

Fig. 5. a - pulmonary capillaries under the influence of 2 weeks hypokinesia; India ink gelatin injection; obj. 40, oc. 7; b - pulmonary arteries under the influence of hypokinesia after 4 weeks (right inferior lobe, rentgenogram); c - pulmonary veins under the influence of hypokinesia for 8 weeks (right inferior lobe, rentgenogram); d - pulmonary capillary bed in 8 week hypokinesia; India ink gelatin injection; obj. 40, oc. 7.

Combined Effect of Hypokinesia and Stress. The following data were obtained in a study of the effect of hypokinesia and hypodynamia in the material from animals subjected to them for periods of 1 to 8 weeks and then the effect of single continuous

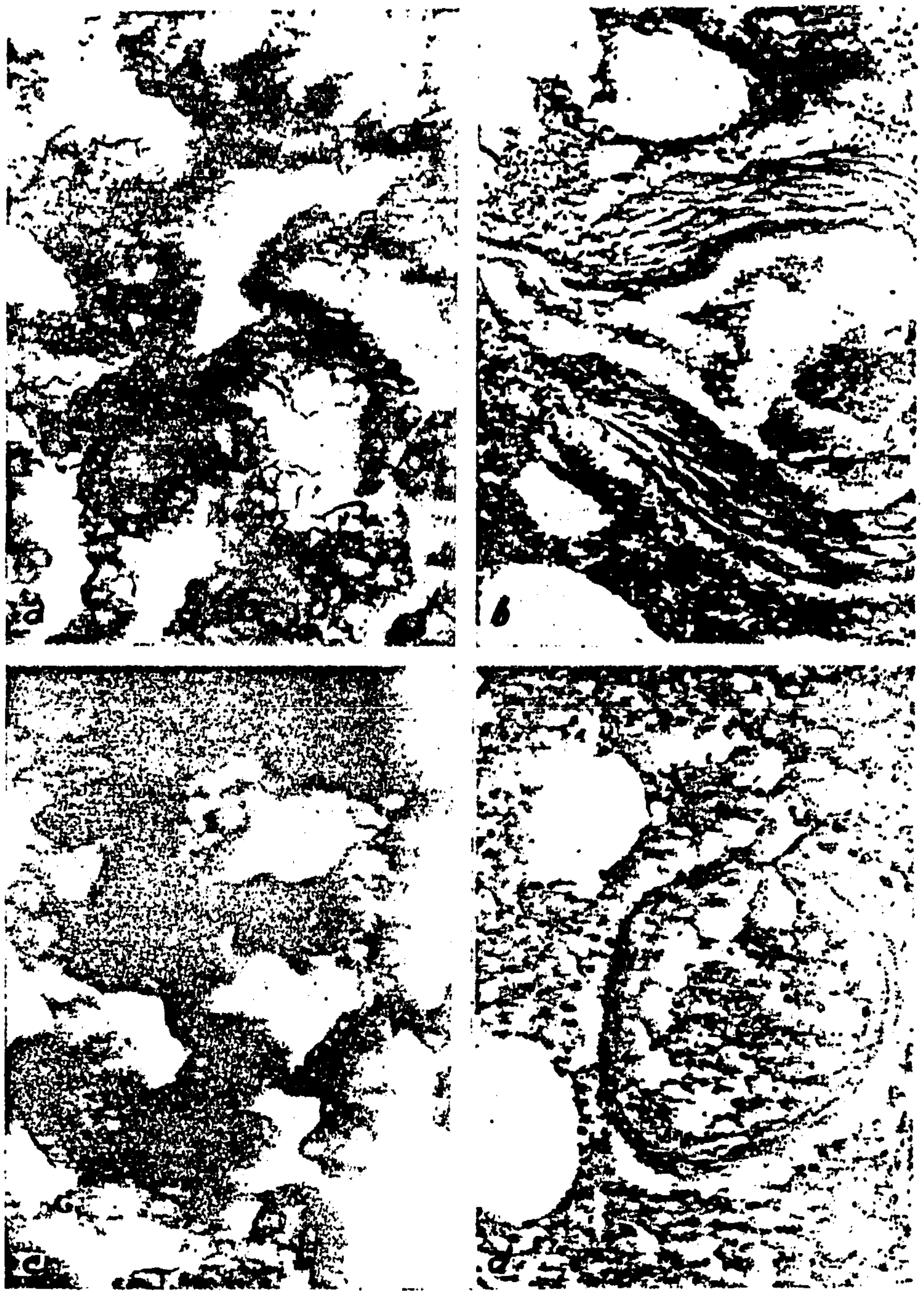

Fig. 6. a – pulmonary capillary bed in the 4th week of hypokinesia with subsequent overloading; injection with Herot's mass; obj. 40, oc. 7; b – deformation of vessel wall in 4th week of hypokinesia with subsequent overloading; microphoto; hematoxylin-eosin; objective 20 , ocular 7; c – pulmonary capillary bed in 8th week of hypokinesia with subsequent overloading; injection with Herot's mass; obj. 40, oc. 7; d – rupture of vascular wall and escape of formal blood elements from vessel; effect of 8th week hypokinesia with subsequent overloading; microphoto; hematoxylin-eosin; obj. 40, oc. 7.

threshold endurable overloads in the chestback direction.

After 1 or 2 weeks hypokinesia and hypodynamia and subsequent overloading the changes in the pulmonary blood vessels differed little from those noted for stress alone and were expressed by the fact, that the distal portions of the arteries of the III and IV orders were constricted, the veins dilated and sinuous. However the changes in the microcirculatory bed were of a much deeper kind: thus the total area of the capillary bed became larger than under the usual influence of overload or of hypokinesia and hypodynamia. The deformation of small arterial and venous vessels under the successive influence of the two factors was more marked, as was clearly shown in the histological preparations.

Four weeks after the influence of hypokinesia and hypodynamia and subsequent effect of overloading the picture of changes in the arterial bed was essentially different from that following the usual action of hypokinesia and hypodynamia for the same time period. If under the influence of hypokinesia and hypodynamia arteries of the III and IV orders were significantly constricted, only some of the arteries of some segments in the ventral portions of the pulmonary lobes were constricted by the successive action of two extreme factors. The veins appeared more dilated and sinuous than in hypokinesia. Especially clear were the deeper changes under the successive influence of the two factors -- in the microcirculatory bed, where they took the form of a larger overall capillary area, deformation of capillary walls and severe dilation (Fig. 6a). The histological preparations show severe dilation and attenuation of the vascular walls, usually from one pole. Sometimes in the area of such protrusions the vascular wall is 2 or 3 times as thick as normal (Fig. 6b). For much longer periods of hypokinesia and hypodynamia (6 and 8 weeks) and subsequent overloading the macroscopic finding is dilation of arteries of the III, IV and V orders in contrast to hypokinesia alone, where the opposite was true. This seems to be explained by the fact, that in hypokinesia and hypodynamia there occurs a disruption of the "intraceptive afferentation" of pulmonary blood vessels (L. A. Nikolayeva, 1971) and as a result of this disruption there is no spasm of the arterial vessels to compensate for the effect of overload. Macroscopically the veins appear more dilated than in the same time periods when there is hypokinesia alone and venous sinuosity is more clearly pronounced. Deformation of the capillary network was greater in comparison with previous time periods (Fig. 6c), all along the capillaries their diameter was very uneven, the capillaries themselves very sinuous, their walls signifi- /89

cantly deformed. In some parts of the pulmonary tissue there are ruptures in the vascular wall and penetration of formed elements of the blood beyond the confines of the vessel (Fig. 6d).

Thus when there are short periods of hypokinesia and hypodynamia with subsequent single, continuous threshold endurable overloading in the chestback direction the changes differ little from the effects of stress alone. It seems that during such shorter periods of hypokinesia and hypodynamia there are no significant disruptions of the "intraceptive afferentation" and one finds no aggravating effect of hypokinesia and hypodynamia.

However when the periods of hypokinesia and hypodynamia are longer (4-6 and especially 8 weeks) the changes are considerably different from those noted for hypokinesia alone without subsequent overload. If with these time periods of hypokinesia alone there is arterial constriction, sometimes so significant as to present foci of spasm, with hypokinesia of the same respective time periods and subsequent stress there is constriction of arteries of the III and IV orders only in some segments but basically the arteries of these orders dilate.

The venous bed is more dilated than with hypokinesia and hypodynamia alone. The wall of the small vessels undergoes greater changes with the successive action of the two factors than for each of them alone. At times the attenuation of the vascular wall was so clearly pronounced, that one could note vascular ruptures, something never noticed when the factors mentioned acted alone. Thus great changes due to hypokinesia and hypodynamia with subsequent overload were noted basically in the microcirculatory bed of the small arteries and veins. It seems that this fact is explained by the fact that resistance to blood flow grows in proportion to reduction in the caliber of the vessels (T. A. Gibradze, 1966; Yu. P. Arkhipchuk, 1967).

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